scan 1 yr earlier prior to treatment showed pulmonary fibrosis suggestive of UIP, with no ground-glass change.

She presented with a 2-week-history of breathlessness after 12 injections (6 weeks) of etanercept, 25 mg twice a week. There was no evidence of infection or heart failure. CXR showed new diffuse reticulonodular shadowing. CT pulmonary angiography (CTPA) confirmed widespread ground-glass change with no evidence of pulmonary embolism.

She was treated with broad spectrum intravenous antibiotics. The etanercept and methotrexate were stopped. Despite this and adding methylprednisolone 500 mg/day for 3 days and changing antibiotics, she developed worsening hypoxia and was intubated. Bronchoalveolar lavage showed no evidence of TB, bacterial or pneumocystis Carinii infection.

She developed metabolic acidosis, progressive renal failure and later cardiac arrhythmias, and despite intensive treatment she died.

Both our patients had pre-existing lung disease, they had enquiry into respiratory symptoms at 3 month intervals and annual CXR prior to the commencement of etanercept and were symptomatically stable. Also, both were on methotrexate and both developed acute respiratory symptoms within 3 and 6 weeks of commencing etanercept, respectively, which culminated in accelerated interstitial lung disease. The patient with COPD presented earlier and recovered quickly with oral steroids alone after discontinuing etanercept and subsequently was able to recommence methotrexate 25 mg/week with adequate joint disease control but no recurrence of respiratory symptoms. The patient with poorer respiratory reserve developed progressive lung disease and died despite aggressive treatment.

Lung disease is a well-known complication of methotrexate, and cases of accelerated methotrexate pneumonitis are also reported within 2–3 doses of infliximab. Our patient who survived restarted methotrexate safely, hence we do not postulate this as a cause [1]. Infliximab has been reported to accelerate lung nodulosis [2, 3], and it has been reported as causing a reversible, biopsy proven, non-caseating granulomatous lung disease in RA [4]. In both our patients, lung biopsies were taken, which may have been the histological change of the respiratory disease. Four further cases of reversible non-caseating granulomatous reaction temporally related to etanercept therapy have also been reported, two of which had previous pulmonary fibrosis [5]. Our two cases demonstrate a little-known complication of etanercept, one of which was fatal. It is noticeable that the patient who died also had rheumatoid lung disease, whilst the patient with COPD survived. Previously reports from the Biologics register [6], and published reports have raised the concern of increased mortality in patients with RA and pre-existing rheumatoid lung disease on azathiaprine when anti-TNF was added [7]. On the basis of these two cases caution needs to be extended to those with pre-existing lung disease, taking methotrexate when etanercept is added, particularly if the lung disease is due to rheumatoid involvement. Extra caution should be taken in patients with rheumatoid lung and poor respiratory reserve. Patients should be prompted to contact the rheumatology department if symptoms of acute breathlessness occur, especially soon after the introduction of etanercept.

Rheumatology	Key message
	• When anti-TNF agents are added, patients with pre-existing rheumatoid lung disease taking methotrexate, are at a risk of acute pulmonary disease progression.

The authors have declared no conflict of interest.

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Accepted 14 February 2006

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Rheumatology 2006;45:1049–1050
doi:10.1093/rheumatology/kel196
Advance Access publication 12 June 2006

Detection of tuberculosis by extensive screening in a patient with rheumatoid arthritis prior to anti-TNF-α therapy

Sir, TNF-α blocking agents have seen increasing use in the treatment of systemic inflammatory diseases such as rheumatoid arthritis (RA). An association of anti-TNF-α agents with the occurrence of mycobacterial infections (MTBI), with a high proportion of extrapulmonary and disseminated cases, has been noted [1, 2]. The article by Horsburgh [3] highlights the public health consequences of MTBI in the setting of anti-TNF-α therapy. It is recommended to exclude MTBI before anti-TNF-α therapy and to give prophylactic isoniazid (INH) if latent MTBI (LMTBI) is suspected [4]. The use of INH in patients >50 yrs is associated with a considerable mortality of 2.3% [5]. In addition, the effectiveness of prophylactic INH therapy is in the range of 25–92%, depending on underlying disease, compliance and type of study [6]. Therefore, we decided to implement a more stringent pre-treatment screening protocol. This encompasses conventional radiographs of the chest in two planes and, if results were questionable, a CT scan. The tuberculin skin test (TST) was performed with two IU injected intracutaneously according to the standard procedure in Switzerland. A reading was taken 48–72 h later, an induration of ≥ 5 mm was considered positive. If negative, the TST was repeated after 7–10 days, with identical criteria applied for reading. If the TST was positive, and/or imaging of the chest suspicious, bronchosopy with lavage and brushings for cultures and nucleic acid amplification tests (NAAT) for mycobacteria, and cultures and NAAT of the urine were performed.

From September 2002 until July 2004, 17 patients with the indication for anti-TNF-α therapy were screened according to this procedure. Six had further examinations, of which three were based on the chest radiograph and three on a positive TST. Among the latter, the following case of LMTBI was detected.

A 76-year-old woman with RA of 14 years duration, who had persistent erosive disease despite various disease-modifying anti-rheumatic drugs (DMARDs), qualified for anti-TNF-α

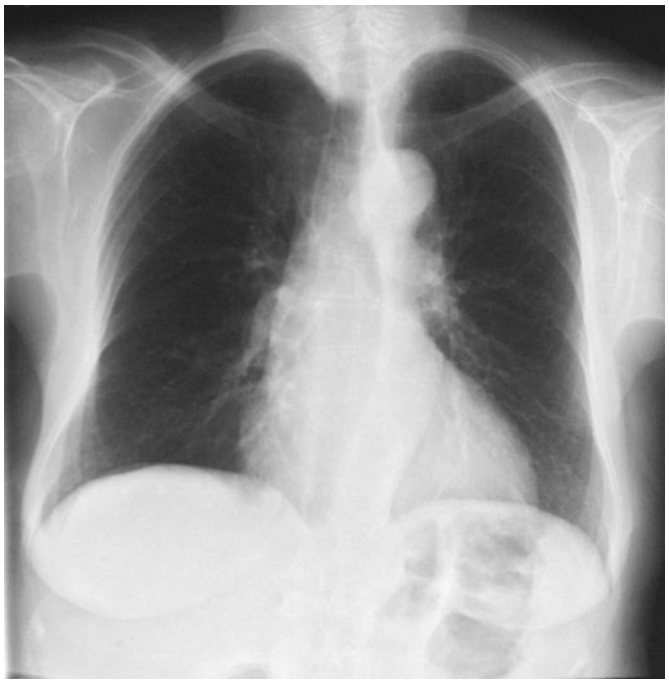

FIG. 1. Radiograph of the chest without signs of latent mycobacterial infection.

treatment. Apart from her age, and the use of DMARDs and low-dose glucocorticosteroids, there were no apparent risk factors for tuberculosis, and clinical signs of an acute or chronic infection were absent. The chest X-ray (Fig. 1) showed no evidence of MTBI, but the TST provoked an induration of 10 mm in diameter. Bronchoscopy was unremarkable and fluorescence microscopy of the specimens and of the urine for mycobacteria were negative. However, cultures and NAATs (Cobas Amplicore MTB) of the tracheobronchial lavage and of the urine identified *Mycobacterium africanum*, which was sensitive to rifampicin (RIF), INH, ethambutol (ETB) and pyrazinamide (PZA). Possible active foci in the chest and abdomen could not be detected by a CT scan of the chest and abdominal ultrasonography. A tuberculostatic regimen with INH 50 mg, PZA 300 mg and RIF 120 mg daily for 2 months followed by INH 100 mg and RIF 150 mg daily for 7 months was given under continued weekly methotrexate and daily low-dose prednisone. Culture and PCR for mycobacteria in the urine turned negative.

This case raises several questions relating to anti-TNF-α therapy. First, how adequate are the current recommendations for the detection of LMTBI? Second, how appropriate is prophylactic INH to prevent the possible activation of LMTBI under anti-TNF-α therapy? Third, what is the cost–benefit ratio of INH prophylaxis in view of potential toxicity and drug resistance? Fourth, are there any differences between the available anti-TNF-α agents? Fifth, will the introduction of T-cell stimulation test with MTB peptides improve the screening procedure for LMTBI [7, 8]? The answers will establish whether more extensive procedures to exclude LMTBI before anti-TNF-α therapy and treatment as necessary are superior to prophylactic INH, with the exposure of patients not needing it, and the potential drawback of resistance. Until these questions are resolved, astute clinical assessment under consideration of the recommendations of national professional societies must guide the handling of this issue.

The authors have declared no conflicts of interest.

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Rheumatology 2006;45:1050–1051
doi:10.1093/rheumatology/kel167
Advance Access publication 22 May 2006

Re: Touch screen computer systems in the rheumatology clinic offer a reliable and user-friendly means of collecting quality-of-life and outcome data from patients with rheumatoid arthritis

SIR, With great interest we read the article by Greenwood *et al.* [1] on the use of touch screen computer systems in the rheumatology clinic. They demonstrated that the use of touch screen questionnaires is a feasible way of data collection in a rheumatology clinic. Another application of interactive computer systems in rheumatology clinics may be the provision of health information [2]. It was found earlier that patients with chronic rheumatic diseases use the Internet primarily to search for information about the disease and its treatment [3]. We would like to share our experience with an interactive patient-information kiosk.

In July 2005, an interactive patient-information kiosk was placed in the waiting area of the rheumatology out-patient clinic. The kiosk had a user-friendly design and provided access to one specific website via a high-speed Internet connection. This website (www.reumanet.nl) contained general information and address/telephone/e-mail directories regarding various regional healthcare services [4] and organizations for patient with rheumatic diseases. Moreover, links to 65 selected other related websites were included. An instruction chart on how to use the kiosk and an information leaflet about the website were present